THE USE OF HYPOTHERMIA IN THE PREVENTION OF PARAPLEGIA FOLLOWING TEMPORARY AORTIC OCCLUSION: EXPERIMENTAL OBSERVATIONS*

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ALTHOUGH recent developments in the surgical treatment of aortic disease by resection have provided a strikingly more favorable outlook for these grave conditions, certain technical factors essential in the performance of the procedure have produced some limitations to the application of this form of therapy.[1, 9, 12-14, 16, 18, 23] This stems primarily from the fact that cross-clamping of the aorta is necessary during the performance of excision of the diseased segment and its replacement by an aortic homograft. In cases in which the lesion is constrictive or obliterative, such as coarctation, this does not constitute a problem owing to the fact that a well-developed collateral circulation already exists. On the other hand, in patients with aneurysmal disease, this becomes a highly important problem, since cross-clamping of the aorta under these circumstances creates potential ischemic hazards of temporary arrest of the circulation to the tissues distal to the point of occlusion. Accordingly, the degree of ischemic damage to organs below the level of aortic occlusion during this critical period usually determines the success or failure of the surgical procedure. The spinal cord distal to the site of aortic occlusion appears to be the most vulnerable tissue to damage from temporary ischemia. Relatively mild ischemic changes in the spinal cord, as may occur in occluding the aorta below the diaphragmatic hiatus, may produce no permanent neurological disturbances. Thus, in our series of forty patients with abdominal aortic aneurysm and twenty-two patients with thrombo-obliterative disease in whom the aorta was occluded for more than one hour, no neurologic sequelae were encountered. On the other hand, occlusion of the descending thoracic aorta below the level of the arch may lead to serious neurological disturbances and even death.

Probably the most important factor determining the extent of damage to the spinal cord under these circumstances is the degree to which collateral vessels have already been developed. With constrictive or obliterative lesions of the thoracic aorta, such as coarctation, a rich supply of collateral circulation is developed which supplies the lower portion of the body. Thus, aortic occlusion in coarctation is seldom a cause for paraplegia.[4, 7, 19] With lesions, however, such as aneurysm of the aorta, in which there is only a slight tendency for collateral circulation to develop, temporary aortic occlusion may cause serious spinal cord changes, paraplegia, and death.[22]

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A number of techniques have already been used experimentally and clinically to provide circulation to the lower segment of aorta during the period of aortic reconstruction. Temporary intubation of the aorta[15, 21] and temporary extra aortic shunts[11, 24] have been used with rather disappointing results in most instances. Another approach to the problem is through reduction of oxygen demand by the central nervous system by lowering body temperature with induced hypothermia.[3, 21] On the basis of clinical and experimental observations with hypothermia for other purposes, there is reason to believe that this may provide an effective solution to the problem.[2, 5, 6, 17, 25, 26] Accordingly, these studies were undertaken to determine the effectiveness of hypothermia in preventing spinal cord damage following temporary arrest of the circulation in the thoracic aorta.

METHOD

Mongrel dogs averaging 7 to 15 kilograms were used in all the experiments. Anesthesia was obtained by intravenous Nembutal, 30 mg. per kilogram of body weight, and artificial respiration was provided by means of endotracheal tube and mechanical insufflator. The aorta was exposed through the third left intercostal space for occlusions just distal to the left subclavian artery. A second small incision, usually through the eighth intercostal space, was used for animals in which occlusion was at the diaphragmatic hiatus as well as proximally. In those animals in which all the intercostal arteries were ligated, exposure was obtained through the bed of the resected seventh rib.

Hypothermia was produced by wrapping the animals in a rubberized blanket through which a refrigerant solution was circulated. Rectal temperatures were reduced to between 75° and 80° F. before the chest was opened. Following completion of the operation, rewarming was accomplished by immersing the animal in a water bath at 110° to 115° F. for fifteen to thirty minutes until rectal temperature had risen to 96° F.

Four groups of experiments were performed, including a control series in each group, with the period of aortic occlusion being one hour for all of the animals:

Group I. The aorta was occluded just distal to the left subclavian artery.

Group II. The aorta was simultaneously occluded just distal to the left subclavian artery and at the level of the diaphragmatic hiatus.

Group III. The aorta was occluded just distal to the left subclavian artery after ligation of the upper two pairs of intercostal arteries.

Group IV. The aorta was occluded just distal to the left subclavian artery after ligation of all of the supradiaphragmatic intercostal arteries.

RESULTS AND DISCUSSION

All the deaths in both the control and hypothermic animals took place within the first six to twelve hours after the experiment, and usually without recovery from anesthesia. It was therefore impossible to determine in these animals whether or not paraplegia existed. Death occurred in most of these animals, both in the control and hypothermic group, in a shocklike state. The mechanism

of death in these animals is not clear. It was thought that overwhelming bacteremia might be a factor, but control of bacteremia by Aureomycin in one group of animals in another experiment did not change the mortality or mode of death.

These studies show that occlusion of the aorta just distal to the left subclavian artery in normothermic animals for a period of one hour produces an appreciable degree of ischemic damage to the spinal cord (Table I). This conforms with observations made by a number of other investigators.[4, 8, 10, 20] It is also apparent from our experiments that interruption of the intercostal vessels enhances the ischemic effect of aortic occlusion upon the cord. Although there was only a slight reduction in over-all mortality following the use of hypothermia in these experiments (Graph I), in the surviving animals there was a striking reduction in the incidence of paraplegia in the hypothermic animals. Thus, the 65 per cent over-all paraplegia rate for the thirty-four surviving control animals was reduced to 0 in the thirty-five surviving hypothermic animals (Graph II). The total influence of hypothermia in reducing the incidence of the ischemic effects of temporary arrest of aortic circulation is rather impressive (Graph III).

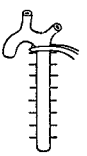
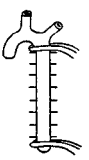
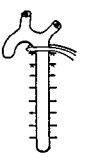
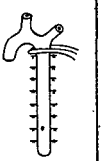
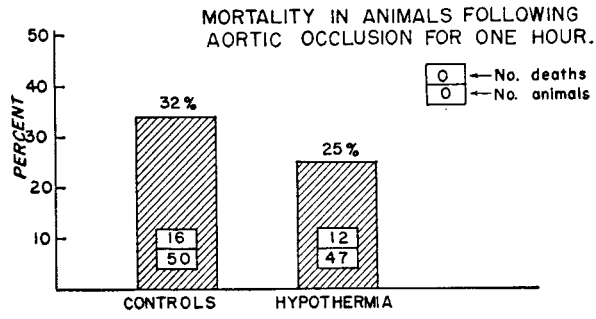
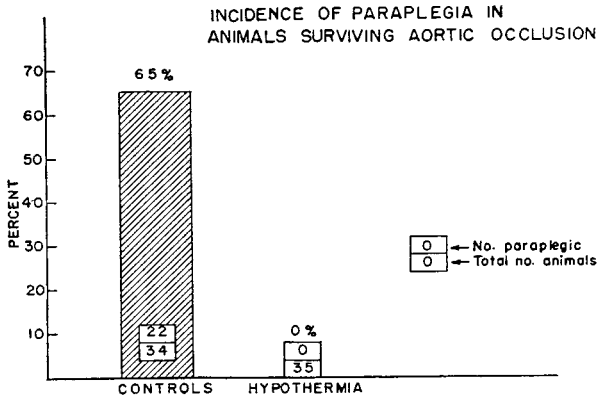
CONTROLS	 Group 1	 Group 2	 Group 3	 Group 4
Total Number	11	18	10	11
Died	1	8	2	5
Paraplegic	3	9	5	5
% Paraplegic	30% (3/10)	90% (9/10)	63% (5/8)	83% (5/6)
HYPOTHERMIC				
Total Number	10	18	10	9
Died	0	8	1	3
Paraplegic	0	0	0	0
%Paraplegic	0% (0/10)	0% (0/10)	0% (0/9)	0% (0/6)

Table I.—Table showing method and results of experiments on the effectiveness of hypothermia in the prevention of paraplegia following temporary aortic occlusion. Four groups of animals were used, cross-clamping the aorta for one hour at the point indicated in the diagram, and with a control series in each group.

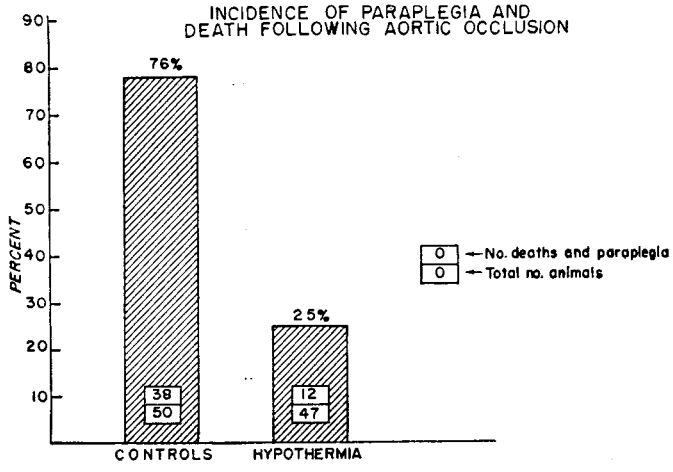
Because cross-clamping of the aorta, as was carried out in the Group II animals, is the type of aortic occlusion that is necessary clinically, additional observations were made in this group of experiments. It was found, as might be expected, that the mean blood pressure in the aorta just distal to the occluding clamp drops precipitously immediately after the occlusion, but within a few minutes begins to rise slowly, reaching a level approximately 20 per cent of the normal within thirty minutes (Fig. 1). On the other hand, in animals



Graph I



Graph II



Graph III

in which the aorta is cross-clamped, as in Group II, the secondary rise in pressure occurs more rapidly and to a much higher degree (Fig. 1). These patterns of pressure response were identical for both normothermic and hypothermic animals. This is further evidence of the importance of collateral blood flow through the intercostal vessels. This may have clinical significance in providing additional means of combatting the ischemic effects of aortic occlusion by deliberately raising the pressure of the aorta distal to the occluding clamp. Further studies along these lines are in progress.

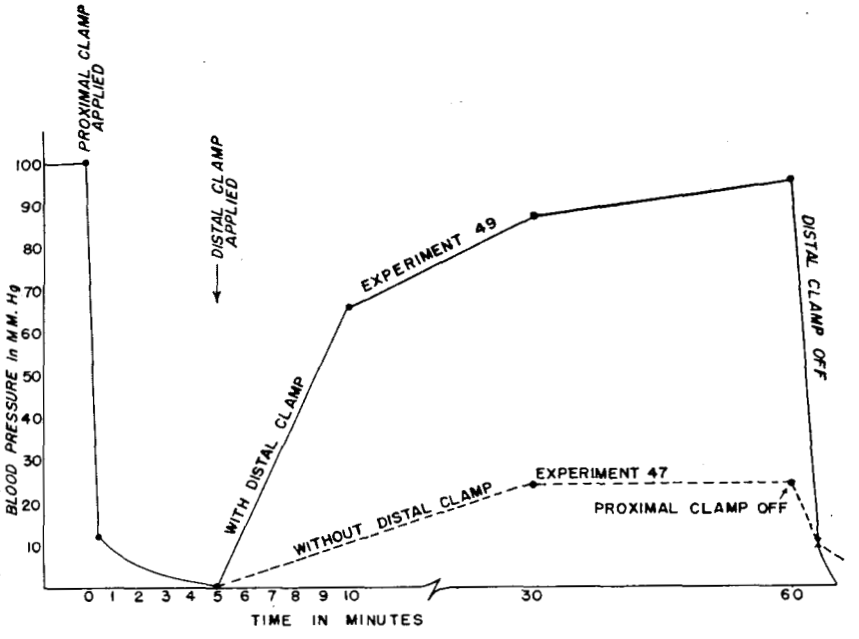


Fig. 1.—Pressure curve made in distal segment of aorta after applying proximal clamp as in Group I animals. Application of distal occluding clamp at level of diaphragm as in Group II caused a striking increase in pressure in the intervening segment of thoracic aorta.

SUMMARY

Studies were undertaken to determine the effectiveness of hypothermia in preventing spinal cord damage following temporary arrest of the circulation in the thoracic aorta. Four groups of experiments were performed with a control series in each group and with the period of aortic occlusion being one hour for all animals. Although the aorta was occluded just distal to the left subclavian artery in all the animals in each group, in certain groups additional measures were used to diminish distal blood flow, such as interruption of intercostal arteries and also the additional occlusion of the aorta at a lower level.

Whereas there was only a slight reduction in over-all mortality following use of hypothermia in these experiments, there was a striking reduction in the incidence of paraplegia. The over-all paraplegia rate for the surviving control animals was 65 per cent, but none of the surviving hypothermic animals de-

veloped paraplegia. On the basis of these experiments it is concluded that hypothermia has a definite protective influence against ischemic damage of the spinal cord following high aortic occlusion.

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